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Fatal Pneumonia Caused by Parainfluenza Type 3 Virus

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MANIFESTATIONS OF MORBIDITY subsequent to parainfluenza virus infections in infants have recently been reported by McCarthy and co-workers.¹ Potential mortality from severe lower respiratory tract infections due to these organisms must also be considered.

From June 1986 through January 1987, parainfluenza virus type 3 was identified in respiratory tract specimens of 29 patients with symptoms of clinical disease at Childrens Hospital of Los Angeles (CHLA). During this interval, two infants younger than 3 months and previously healthy died as a result of severe injury of the lower respiratory tract presumably by this viral agent.

Reports of Cases

Case 1

The patient, a 6-week-old female infant, presented to the CHLA emergency department with rhinorrhea, cough, and congestion for one week. She was noted to be in moderate respiratory distress, and a chest x-ray film showed bilateral pneumonitis. The patient then became apneic, requiring the insertion of an endotracheal tube. Despite increased ventilatory support and the parenteral administration of ampicillin and gentamicin sulfate, she died of respiratory failure on the 15th day. Cultures of specimens of blood, cerebrospinal fluid, urine, stool, and tracheal aspirate were reported as negative for bacteria. Cultures for respiratory syncytial virus, cytomegalovirus, and *Chlamydia trachomatis* were also negative. An endotracheal tube aspirate obtained on the second hospital day and inoculated into rhesus monkey kidney cells yielded after 14 days parainfluenza virus type 3.

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A request for a postmortem examination was denied by the family.

Case 2

The patient, a 3-month-old female infant, was seen in the CHLA emergency department with fever and respiratory distress. A septic workup was done and therapy begun with ampicillin and chloramphenicol sodium succinate. A chest x-ray film showed bilateral interstitial pneumonitis. On the fourth hospital day, increasing respiratory distress developed, and the patient required the insertion of an endotracheal tube. Despite respiratory support, her pulmonary status deteriorated, and she died on the seventh hospital day. Cultures of specimens of blood, cerebrospinal fluid, urine, and endotracheal aspirate were negative for bacteria. Specimens from nasopharynx and endotracheal tube obtained on the fourth hospital day yielded parainfluenza virus type 3, twelve days after being inoculated into rhesus monkey kidney cells.

On postmortem examination, there was extensive interstitial pneumonitis, and cultures of specimens of lung were positive for parainfluenza type 3. In addition, acute bronchopneumonia with lung cultures positive for *Pseudomonas aeruginosa* was identified. A histopathologic evaluation did not suggest the presence of a congenital immune deficiency or an underlying cardiac abnormality.

Discussion

While parainfluenza virus type 3 is recognized as an important cause of lower respiratory tract disease, there were no deaths in the series reported by McCarthy and associates.¹ Our report indicates that parainfluenza type 3 can cause fatal lower respiratory tract disease in previously healthy infants. While the presence of an immunologic, cardiac, or pulmonary disorder cannot be entirely excluded in case 1, a complete postmortem examination in case 2 did not reveal such preexisting abnormalities.

Although the sensitivity of rapid diagnostic techniques to detect respiratory syncytial virus is considered to be at least 90%, a recent report indicates that rapid diagnostic tests to detect parainfluenza type 3 virus show poor sensitivity.² In addition, isolating parainfluenza viruses may require an average of more than six days,³ which lessens the clinical impact of identifying the agent. A modification of cell culture screening methods⁴ may substantially shorten the detection time.

The optimal care of infants with severe lower respiratory tract infections caused by parainfluenza viruses includes aggressive supportive measures. The availability of diagnostic techniques for detecting parainfluenza virus with enhanced sensitivity and acceptable degrees of specificity would permit the consideration of additional therapeutic methods such as the use of ribavirin⁵ and the intravenous administration of immune globulin.⁶

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